CLINICAL IMAGE

Cardiac involvement in hypereosinophilic syndrome

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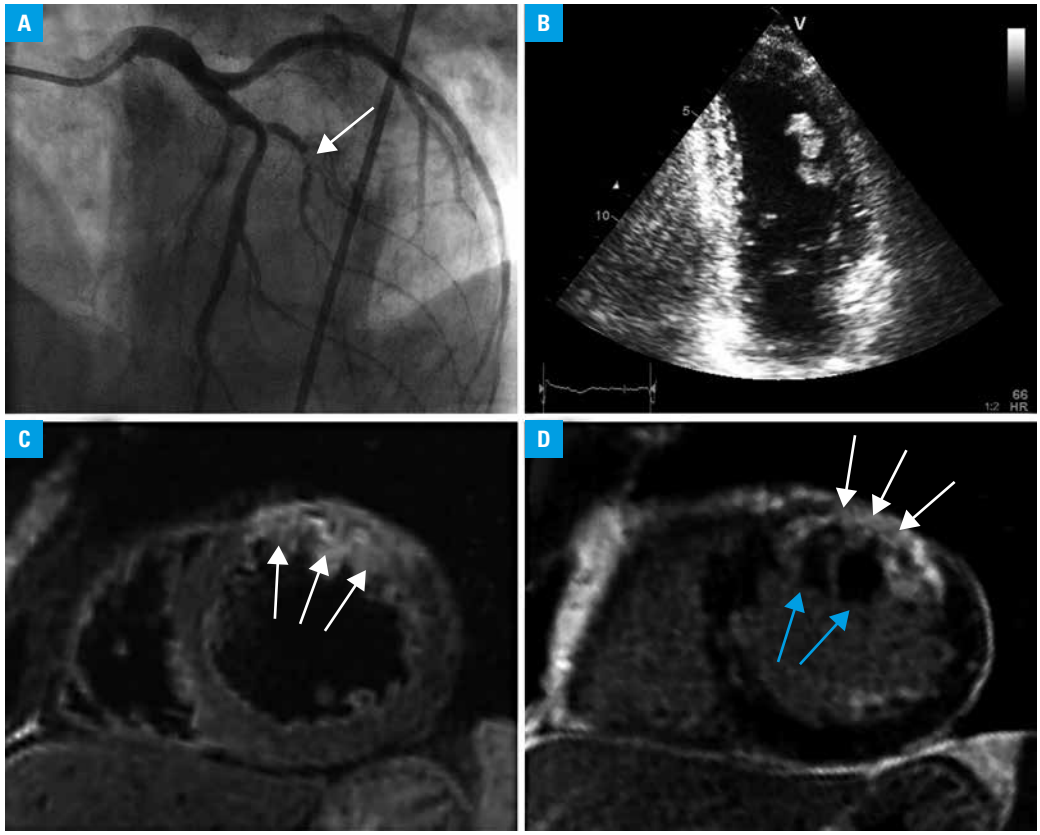

FIGURE 1 Multimodality imaging in a 48-year-old man with hypereosinophilic syndrome and recurrent non-ST-segment elevation myocardial infarction (NSTEMI). Invasive coronary angiography (**A**) revealed in-stent thrombosis in the first diagonal branch of the left anterior descending coronary artery (arrow). Transthoracic echocardiography (**B**) performed 14 days after NSTEMI showed 2 mobile thrombi pedunculated to an akinetic mid anterior segment. Cardiac magnetic resonance confirmed akinesis of the mid anteroseptal and anterior segments and demonstrated myocardial edema (**C**) in the mid anterior segment (arrows) using the STIR sequence. Postcontrast magnetic resonance imaging (**D**) revealed: a) transmural late gadolinium enhancement (LGE) in the mid anterior and anteroseptal segments (white arrows) representing myocardial infarction, and b) linear intramural LGE in the mid septal segment, subendocardial LGE in the mid septal and mid lateral segments, and subepicardial LGE in the mid lateral segment, representing nonischemic injury. Two mobile thrombi pedunculated to an akinetic mid anterior segment (blue arrows) were depicted.

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A 48-year-old man with hypereosinophilic syndrome (HES) of unknown etiology was admitted with recurrent non-ST-segment elevation myocardial infarction (NSTEMI) and the cardiac troponin I levels of 18.2 mg/l. A month earlier, due to the first episode of NSTEMI, the first diagonal branch of the left anterior descending coronary artery was stented, followed by administration of 75 mg aspirin and 75 mg clopidogrel. Due to exacerbation of HES (erythrocyte sedimentation rate, 130 mm/h; C-reactive protein, 96 mg/l; immunoglobulin E, 3360 U/l; leukocytosis, 27,700/μl; peripheral blood eosinophilia, 8642/μl), methylprednisolone (1 mg/kg) was introduced 2 weeks before admission. Two days after admission, coronary angiography showed in-stent thrombosis in the first diagonal branch, successfully reopened during the procedure (**FIGURE 1A**). Two weeks later, transthoracic echocardiography (TTE) showed left ventricular systolic (ejection fraction, 45%) and diastolic dysfunction (E/A, 0.5; deceleration time, 444 ms) as well as 2 mobile thrombi pedunculated to the akinetic mid anterior segment (**FIGURE 1B**). Cardiac magnetic resonance (CMR) confirmed the presence of thrombi as well as myocardial edema (**FIGURE 1C**) and transmural late gadolinum enhancement (LGE) in the mid anterior and anteroseptal wall with the subendocardial no-reflow phenomenon (**FIGURE 1D**). Moreover, CMR demonstrated nonischemic LGE lesions: 1) intramurally in the mid septal, 2) subendocardially in the mid septal and mid lateral, and 3) subepicardially in the mid lateral segments. Enoxaparin (2×1 mg/kg) and methylprednisolone (3 daily pulses of 1000 mg IV, followed by 1 mg/kg) were administered in addition to dual antiplatelet therapy (aspirin and clopidogrel). Seven days later, TTE showed dissolution of one thrombus and a significant reduction in the size of the other thrombus. A week later, both thrombi completely disappeared.

HES is characterized by marked persistent eosinophilia of unknown origin and by eosinophil-mediated organ damage. Cardiac involvement, which affects more than 50% of the patients with HES, is a major cause of morbidity and mortality.[1,2] Cardiac damage progresses in 3 stages. An acute myocardial necrosis is followed by thrombus formation and ultimately progresses to endomyocardial fibrosis, resulting in restrictive cardiomiopathy.[3] Thromboembolic complications are frequent and are associated with poor outcomes.[3]

Our report demonstrates the utility of TTE and CMR in HES. CMR, unlike TTE, allows to identify heart damage in the first stage of the disease. The technique enables to visualize LGE lesions, which may represent inflammation or fibrosis.[4] However, the differentiation between inflammation and fibrosis may be difficult, and our case showed that small LGE areas cannot be unequivocally attributed to one of these processes. LGE pattern, location, transmural distribution, and relation to myocardial coronary artery supply helps distinguish ischemic from nonischemic myocardial injuries.[4] In our patient, CMR delineated transmural myocardial infarction in the region supplied by the first diagonal branch, identified nonischemic myocardial injury in the mid septal and lateral segments, and excluded significant endomyocardial fibrosis. TTE is definitely the first-line technique to detect intracardiac thrombi. Therefore, to exclude intracardiac thrombus in HES complicated by acute myocardial infarction, sequential TTE should be performed, and CMR, as a superior technique, might be recommended in high-risk patients.

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